induces the expression of the HO-1 gene through phosphorylation of the nuclear proteins which bind to the delta 12-PGJ2-responsive element.

FILE 'CAPLUS' ENTERED AT 11:05:36 ON 24 JUL 2007
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FILE 'MEDLINE' ENTERED AT 11:05:36 ON 24 JUL 2007

FILE 'BIOSIS' ENTERED AT 11:05:36 ON 24 JUL 2007
Copyright (c) 2007 The Thomson Corporation

=> s transplant
L1 244960 TRANSPLANT

=> s graft
L2 428898 GRAFT

=> s rhein/cn
'CN' IS NOT A VALID FIELD CODE
L3 434 RHEIN/CN

=> fil reg

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	5.77	5.98

FILE 'REGISTRY' ENTERED AT 11:06:22 ON 24 JUL 2007
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Property values tagged with IC are from the ZIC/VINITI data file provided by InfoChem.

STRUCTURE FILE UPDATES: 23 JUL 2007 HIGHEST RN 943188-87-2
DICTIONARY FILE UPDATES: 23 JUL 2007 HIGHEST RN 943188-87-2

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TSCA INFORMATION NOW CURRENT THROUGH December 2, 2006

Please note that search-term pricing does apply when conducting SmartSELECT searches.

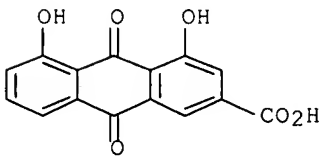
REGISTRY includes numerically searchable data for experimental and predicted properties as well as tags indicating availability of experimental property data in the original document. For information on property searching in REGISTRY, refer to:

http://www.cas.org/support/stngen/stndoc/properties.html

=> s rhein/cn
L4 1 RHEIN/CN

=> d l4

L4 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2007 ACS on STN
RN 478-43-3 REGISTRY
ED Entered STN: 16 Nov 1984
CN 2-Anthracenecarboxylic acid, 9,10-dihydro-4,5-dihydroxy-9,10-dioxo- (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN 2-Anthraquinonecarboxylic acid, 4,5-dihydroxy- (6CI)
CN 2-Anthroic acid, 9,10-dihydro-4,5-dihydroxy-9,10-dioxo- (8CI)
OTHER NAMES:
CN 1,8-Dihydroxy-3-carboxyanthraquinone
CN 1,8-Dihydroxyanthraquinone-3-carboxylic acid
CN 4,5-Dihydroxy-2-anthraquinonecarboxylic acid
CN Cassic acid
CN Chrysazin-3-carboxylic acid
CN Monorhein
CN NSC 38629
CN Rheic acid
CN **Rhein**
CN Rheinic acid
CN Rhubarb yellow
MF C15 H8 O6
CI COM
LC STN Files: ANABSTR, BEILSTEIN*, BIOSIS, BIOTECHNO, CA, CAOLD, CAPLUS, CASREACT, CHEMCATS, CHEMINFORMRX, CHEMLIST, CSCHEM, DDFU, DRUGU, EMBASE, IPA, MEDLINE, MRCK*, NAPRALERT, PS, RTECS*, SYNTHLINE, TOXCENTER, USPAT2, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**
(**Enter CHEMLIST File for up-to-date regulatory information)



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

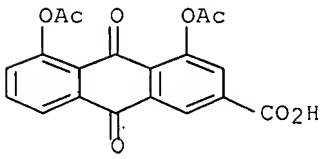
766 REFERENCES IN FILE CA (1907 TO DATE)
25 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
776 REFERENCES IN FILE CAPLUS (1907 TO DATE)
20 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

=> s diacerein/cn
L5 1 DIACEREIN/CN

=> d l5

L5 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2007 ACS on STN
RN 13739-02-1 REGISTRY
ED Entered STN: 16 Nov 1984

CN 2-Anthracenecarboxylic acid, 4,5-bis(acetyloxy)-9,10-dihydro-9,10-dioxo-
(CA INDEX NAME)
OTHER CA INDEX NAMES:
CN 2-Anthroic acid, 9,10-dihydro-4,5-dihydroxy-9,10-dioxo-, diacetate (8CI)
CN Rhein, diacetate (6CI)
OTHER NAMES:
CN 1,8-Diacetoxy-3-carboxyanthraquinone
CN 4,5-Diacetoxyanthraquinone-2-carboxylic acid
CN 4,5-Diacetylrhein
CN Artrodar
CN DAR
CN **Diacerein**
CN Diacerhein
CN Diacetylrhein
CN Fisiodar
CN SF 277
MF C19 H12 O8
CI COM
LC STN Files: ADISINSIGHT, ADISNEWS, AGRICOLA, BEILSTEIN*, BIOSIS,
BIOTECHNO, CA, CAOLD, CAPLUS, CASREACT, CHEMCATS, CHEMLIST, CIN, CSCHEM,
DDFU, DRUGU, EMBASE, IMSDRUGNEWS, IMSPATENTS, IMSRESEARCH, IPA, MEDLINE,
MRCK*, NAPRALERT, PHAR, PROMT, PROUSDDR, PS, RTECS*, SYNTHLINE,
TOXCENTER, USAN, USPAT2, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**, WHO
(**Enter CHEMLIST File for up-to-date regulatory information)



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

147 REFERENCES IN FILE CA (1907 TO DATE)
3 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
147 REFERENCES IN FILE CAPLUS (1907 TO DATE)
3 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

=> fil caplus medline biosis

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	14.25	20.23

FILE 'CAPLUS' ENTERED AT 11:06:54 ON 24 JUL 2007
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FILE 'MEDLINE' ENTERED AT 11:06:54 ON 24 JUL 2007

FILE 'BIOSIS' ENTERED AT 11:06:54 ON 24 JUL 2007

=> d hist

(FILE 'HOME' ENTERED AT 11:05:15 ON 24 JUL 2007)

FILE 'CAPLUS, MEDLINE, BIOSIS' ENTERED AT 11:05:36 ON 24 JUL 2007
L1 244960 S TRANSPLANT
L2 428898 S GRAFT
L3 434 S RHEIN/CN

FILE 'REGISTRY' ENTERED AT 11:06:22 ON 24 JUL 2007
L4 1 S RHEIN/CN
L5 1 S DIACEREIN/CN

FILE 'CAPLUS, MEDLINE, BIOSIS' ENTERED AT 11:06:54 ON 24 JUL 2007

=> s (l4 or rhein or l5 or diacerein) and (l1 or l2)
L6 9 (L4 OR RHEIN OR L5 OR DIACEREIN) AND (L1 OR L2)

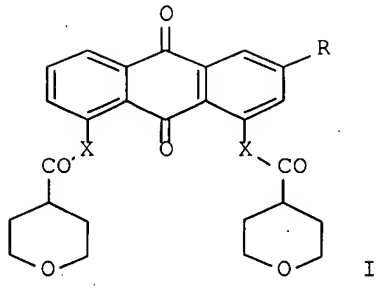
=> d ibib abs 1-9

L6 ANSWER 1 OF 9 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2007:175514 CAPLUS Full-text
DOCUMENT NUMBER: 146:251663
TITLE: Preparation of dihydroxyanthraquinones for therapeutic use as anti-inflammatory agents
INVENTOR(S): Baxter, Andrew Douglas; Walmsley, Andrea
PATENT ASSIGNEE(S): Sosei R & D Ltd., UK
SOURCE: PCT Int. Appl., 22pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2007017695	A2	20070215	WO 2006-GB2999	20060810
WO 2007017695	A3	20070518		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA

PRIORITY APPLN. INFO.: GB 2005-16469 A 20050810
OTHER SOURCE(S): MARPAT 146:251663
GI



I

AB **Rhein** related dihydroxyanthraquinones, such as I [R = morpholin-4-ylcarbonyl, NHEt, CO2H, CO2Et, CO2(CH2)2OMe, X = O; R = CN, tetrazol-5-yl, X = CH2], were prepared for use in pharmaceutical compns. useful in the treatment of inflammatory and autoimmune diseases and conditions associated with T-cell proliferation or that are mediated by pro-inflammatory cytokines, particularly IL-1β or IL-18. These autoimmune and inflammatory diseases and conditions may include chronic degenerative disease, such as rheumatoid arthritis, osteoarthritis or osteoporosis, chronic demyelinating disease, such as multiple sclerosis, respiratory disease, such as asthma or chronic obstructive pulmonary disease (COPD), inflammatory bowel disease (IBD) such as ulcerative colitis or Crohn's disease, dermatol. conditions, such as psoriasis, scleroderma or atopic dermatitis, dental diseases, such as periodontal disease or gingivitis, diabetic nephropathy, lupus nephritis, IgA nephropathy, glomerulonephritis, systemic lupus erythematosus (SLE), **graft** vs. host disease, cancer, diabetic retinopathy, age-related macular degeneration, and conditions characterized by angiogenesis. Thus, dihydroxyanthraquinone I (R = morpholin-4-ylcarbonyl, X = O) was prepared via an amidation reaction with 87% yield of the corresponding carboxylic acid I (R = CO2H, X = O) with morpholine using EDCI and HOBt in CH2Cl2. The prepared compds. were assayed for their effect on serum TNFα and IL-10 levels using the LPS mouse assay, and they were assayed for anti-inflammatory using the Carrageenan paw edema assay in rats.

L6 ANSWER 2 OF 9 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2005:1291308 CAPLUS Full-text
DOCUMENT NUMBER: 144:252074
TITLE: Heat-shock proteins and their role in chondrocyte protection, an application for autologous transplantation
AUTHOR(S): Sawatzky, D. A.; Foster, R.; Seed, M. P.; Willoughby, D. A.
CORPORATE SOURCE: Experimental Pathology Group, William Harvey Research Institute, Saint Bartholomew's and the Royal London School of Medicine and Dentistry, Queen Mary and Westfield College, London, EC1M 6BQ, UK
SOURCE: Inflammopharmacology (2005), 12(5-6), 569-589
CODEN: IAOAES; ISSN: 0925-4692
PUBLISHER: VSP
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Articular cartilage injury presents a unique therapeutic challenge. As cartilage possesses no blood or nerve supply of its own it has a particular susceptibility to early injury and a poor capacity for self-repair. Treatment options are limited and injury can eventually lead to osteoarthritis. Autologous chondrocyte transplantation is an exciting therapeutic development, but despite initial encouraging results, **graft** failure and formation of fibro-

as opposed to hyaline cartilage remain problematic. Bleeding is an inevitable consequence of surgery, and blood-induced cartilage damage is well documented. It is hypothesised here that protecting chondrocytes against blood could significantly improve results. Heat-shock protein induction may confer chondroprotection. The expression of heat-shock proteins in human chondrocytes and rat femoral head cartilage following heat shock was analyzed by Western blotting, and red-blood-cell-induced chondrocyte death was assessed by cell viability and apoptosis by flow cytometry. We demonstrate that heat-shock induced expression of heat-shock protein 70 (HSP70) (rat and human) and HSP32 (human). Blood and blood products reduced rat cartilage proteoglycan synthesis and human chondrocyte viability, and induced human chondrocyte apoptosis at concns. considerably lower than those reported previously. The induction of HSP70 in rat cartilage was ineffective in reducing chondrocyte death in the absence or presence of red blood cells or red cell products. Heat shock to human chondrocytes reduced low levels of apoptosis (<20%) and cell death induced by low levels of blood products, but not higher levels. Induction of HSP32 with diacetylrhein appeared to be more effective and may hold greater promise. Blood has potent adverse effects on chondrocytes and the induction and chondroprotective effects of heat-shock proteins could be applied to increase the initial success of implanted chondrocytes improving the outcome of autologous chondrocyte transplantation.

REFERENCE COUNT: 21 THERE ARE 21 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 3 OF 9 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2004:711494 CAPLUS Full-text

DOCUMENT NUMBER: 141:225524

TITLE: Preparation of 1,8-dihydroxyanthraquinone-6-carboxamide derivatives as inhibitors of T-cell proliferation for treatment of autoimmune or inflammatory conditions

INVENTOR(S): Bannister, Robin Mark; Baxter, Andrew Douglas; Cooper, Nicola; Brew, John

PATENT ASSIGNEE(S): Arakis Ltd., UK

SOURCE: Brit. UK Pat. Appl., 17 pp.
CODEN: BAXXDU

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
GB 2398780	A	20040901	GB 2003-4395	20030226
PRIORITY APPLN. INFO.:			GB 2003-4395	20030226

OTHER SOURCE(S): CASREACT 141:225524; MARPAT 141:225524

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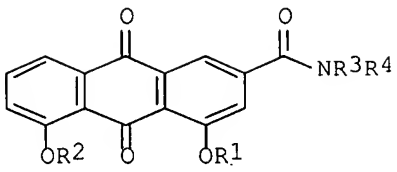

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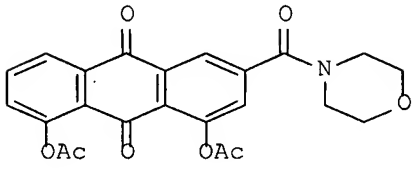


II

AB Title compds. represented by the formula I [wherein R1, R2 = independently H, alkyl, COR5; R3 = H or alkyl; R4 = (un)substituted (cyclo)alkyl, (hetero)aryl; NR3R4 = (un)substituted heterocyclic ring; R5 = alkyl or (hetero)aryl; and pharmaceutically acceptable salts, solvates or hydrates thereof] were prepared as inhibitors of T-cell proliferation (no data). For example, chlorination of 4,5-diacetoxy-9,10-dioxoanthracene-2-carboxylic acid with thionyl chloride and followed by reaction with morpholine, gave II. Thus, I and their pharmaceutical compns. are useful for the treatment of an autoimmune or inflammatory conditions including a chronic degenerative disease (such as rheumatoid arthritis, osteoarthritis or osteoporosis), a chronic demyelinating disease (such as multiple sclerosis), a respiratory disease (such as asthma or allergic rhinitis or chronic obstructive pulmonary disease [COPD]), an inflammatory bowel disease [IBD] (such as ulcerative colitis or Crohn's disease), a dermatol. condition (such as psoriasis, scleroderma or atopic dermatitis), a dental disease (such as periodontal disease or gingivitis), diabetic nephropathy, lupus nephritis, IgA nephropathy, glomerulonephritis, systemic lupus erythematosus (SLE) or **graft** vs. host disease (no data).

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 4 OF 9 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 2004:711493 CAPLUS Full-text

DOCUMENT NUMBER: 141:225167

TITLE: Preparation of 1,8-dihydroxyanthraquinone-6-carboxamide derivatives as modulators of Il-10 production for treatment of autoimmune or inflammatory conditions

INVENTOR(S): Bannister, Robin Mark; Baxter, Andrew Douglas; Cooper, Nicola; Brew, John

PATENT ASSIGNEE(S): Arakis Limited, UK

SOURCE: Brit. UK Pat. Appl., 15 pp.
CODEN: BAXXDU

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

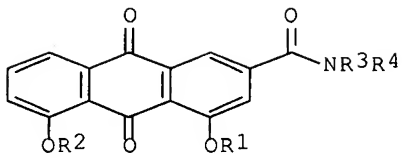
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
GB 2398779	A	20040901	GB 2003-4394	20030226

PRIORITY APPLN. INFO.: GB 2003-4394 20030226

OTHER SOURCE(S): MARPAT 141:225167

GI



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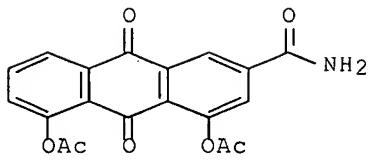

II

AB Title compds. represented by the formula I [wherein R1, R2 = independently H, alkyl, COR5; R3, R4 = independently H or alkyl; R5 = alkyl or (hetero)aryl; and pharmaceutically acceptable salts, solvates or hydrates thereof] were prepared as modulators of Il-10 production (no data). For example, chlorination of 4,5-diacetoxy-9,10-dioxoanthracene-2-carboxylic acid with thionyl chloride and followed by reaction with ammonia, gave II. Thus, I and their pharmaceutical compns. are useful for the treatment of an autoimmune or inflammatory conditions including a chronic degenerative disease (such as rheumatoid arthritis, osteoarthritis or osteoporosis), a chronic demyelinating disease (such as multiple sclerosis), a respiratory disease (such as asthma or allergic rhinitis or chronic obstructive pulmonary disease [COPD]), an inflammatory bowel disease [IBD] (such as ulcerative colitis or Crohn's disease), a dermatol. condition (such as psoriasis, scleroderma or atopic dermatitis), a dental disease (such as periodontal disease or gingivitis), diabetic nephropathy, lupus nephritis, IgA nephropathy, glomerulonephritis, systemic lupus erythematosus (SLE) or **graft** vs. host disease (no data). These carboxamide derivs. are capable of enhancing IL-10 production and inhibiting T-cell proliferation in assays (no data).

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 5 OF 9 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 2004:80181 CAPLUS Full-text
DOCUMENT NUMBER: 140:133914
TITLE: Use of a **rhein** for the preparation of a drug for the treatment of chronic inflammation, and the prevention and the treatment of organ and tissue **transplant** rejection
INVENTOR(S): Charbit, Suzy; Ficheux, Herve; Provvedini, Diego; Schutze, Francois
PATENT ASSIGNEE(S): Negma-Lerads, Fr.
SOURCE: Fr. Demande, 28 pp.
CODEN: FRXXBL
DOCUMENT TYPE: Patent
LANGUAGE: French
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
FR 2842738	A1	20040130	FR 2002-9340	20020723
FR 2842738	B1	20060210		
CA 2493074	A1	20040205	CA 2003-2493074	20030718
WO 2004010990	A1	20040205	WO 2003-FR2286	20030718

W: AU, BR, CA, CN, DZ, IL, IN, JP, KR, MA, MX, NO, NZ, PL, RU, TN, US, VN, ZA
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE,

IT, LU, MC, NL, PT, RO, SE, SI, SK, TR

AU 2003269037	A1	20040216	AU 2003-269037	20030718
EP 1523312	A1	20050420	EP 2003-750824	20030718
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, SK				
JP 2005538098	T	20051215	JP 2004-523858	20030718
MX 2005PA00904	A	20050323	MX 2005-PA904	20050121
US 2006058392	A1	20060316	US 2005-522035	20050929
PRIORITY APPLN. INFO.:			FR 2002-9340 A	20020723
			WO 2003-FR2286 W	20030718

OTHER SOURCE(S): MARPAT 140:133914

AB **Rhein**, diacerhein, their salts and esters can be used for the treatment of chronic inflammations or prevention and treatment of the **transplant** rejections of tissues and organs. Efficacy of diacerhein and **rhein** in prevention of **transplant** rejections are shown in rats.

REFERENCE COUNT: 10 THERE ARE 10 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 6 OF 9 CAPLUS COPYRIGHT 2007 ACS on STN

ACCESSION NUMBER: 1968:473886 CAPLUS Full-text
DOCUMENT NUMBER: 69:73886
TITLE: **Rhein**: an inhibitor of mitochondrial oxidations
AUTHOR(S): Kean, E. A.
CORPORATE SOURCE: Univ. West Indies, Kingston, Jamaica
SOURCE: Archives of Biochemistry and Biophysics (1968), 127(1-3), 528-33
CODEN: ABBIA4; ISSN: 0003-9861
DOCUMENT TYPE: Journal
LANGUAGE: English

AB **Rhein**, an anthraquinone from plants of the genus Cassia, is an inhibitor of electron transport. Test systems included homogenates and mitochondria, both intact and disrupted, from rat liver and kidney. The compound effectively blocked DPN-linked oxidns., while leaving oxidation of succinate relatively unaffected. The action was attributable neither to effects on oxidizable groups such as SH not to the establishment of any pathway bypassing the normal H and electron carriers. Only slight uncoupling of oxidative phosphorylation was observed. The evidence indicated inhibition occurring within the DPNH-cytochrome c reductase complex at a point located on the substrate side of coenzyme Q. 24 references.

L6 ANSWER 7 OF 9 MEDLINE on STN

ACCESSION NUMBER: 2005582541 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 16259722
TITLE: Heat-shock proteins and their role in chondrocyte protection, an application for autologous transplantation.
AUTHOR: Sawatzky D A; Foster R; Seed M P; Willoughby D A
CORPORATE SOURCE: Experimental Pathology Group, William Harvey Research Institute, Saint Bartholomew's and the Royal London School of Medicine and Dentistry, Queen Mary and Westfield College, Charterhouse Square, London, EC1M 6BQ, UK.
SOURCE: Inflammopharmacology, (2005) Vol. 12, No. 5-6, pp. 569-89. Journal code: 9112626. ISSN: 0925-4692.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200705

ENTRY DATE: Entered STN: 3 Nov 2005
Last Updated on STN: 16 Dec 2005
Entered Medline: 29 May 2007

AB Articular cartilage injury presents a unique therapeutic challenge. As cartilage possesses no blood or nerve supply of its own it has a particular susceptibility to early injury and a poor capacity for self-repair. Treatment options are limited and injury can eventually lead to osteoarthritis. Autologous chondrocyte transplantation is an exciting therapeutic development, but despite initial encouraging results, **graft** failure and formation of fibro- as opposed to hyaline cartilage remain problematic. Bleeding is an inevitable consequence of surgery, and blood-induced cartilage damage is well documented. It is hypothesised here that protecting chondrocytes against blood could significantly improve results. Heat-shock protein induction may confer chondroprotection. The expression of heat-shock proteins in human chondrocytes and rat femoral head cartilage following heat shock was analysed by Western blotting, and red-blood-cell-induced chondrocyte death was assessed by cell viability and apoptosis by flow cytometry. We demonstrate that heat-shock induced expression of heat-shock protein 70 (HSP70) (rat and human) and HSP32 (human). Blood and blood products reduced rat cartilage proteoglycan synthesis and human chondrocyte viability, and induced human chondrocyte apoptosis at concentrations considerably lower than those reported previously. The induction of HSP70 in rat cartilage was ineffective in reducing chondrocyte death in the absence or presence of red blood cells or red cell products. Heat shock to human chondrocytes reduced low levels of apoptosis (<20%) and cell death induced by low levels of blood products, but not higher levels. Induction of HSP32 with diacetylrhein appeared to be more effective and may hold greater promise. Blood has potent adverse effects on chondrocytes and the induction and chondroprotective effects of heat-shock proteins could be applied to increase the initial success of implanted chondrocytes improving the outcome of autologous chondrocyte transplantation.

L6 ANSWER 8 OF 9 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 2006:61820 BIOSIS Full-text
DOCUMENT NUMBER: PREV200600051638
TITLE: Heat-shock proteins and their role in chondrocyte protection, an application for autologous transplantation.
AUTHOR(S): Sawatzky, D. A.; Foster, R.; Seed, M. P. [Reprint Author]; Willoughby, D. A.
CORPORATE SOURCE: Univ London St Bartholomews Hosp Med Coll, William Harvey Res Inst, Expt Pathol Grp, London EC1M 6BQ, UK
m.p.seed@qmul.ac.uk
SOURCE: Inflammopharmacology, (2005) Vol. 12, No. 5-6, pp. 569-589. ISSN: 0925-4692.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 4 Jan 2006
Last Updated on STN: 4 Jan 2006

AB Articular cartilage injury presents a unique therapeutic challenge. As cartilage possesses no blood or nerve supply of its own it has a particular susceptibility to early injury and a poor capacity for self-repair. Treatment options are limited and injury can eventually lead to osteoarthritis. Autologous chondrocyte transplantation is an exciting therapeutic development, but despite initial encouraging results, **graft** failure and formation of fibro- as opposed to hyaline cartilage remain problematic. Bleeding is an inevitable consequence of surgery, and blood-induced cartilage damage is well documented. It is hypothesised here that protecting chondrocytes against blood could significantly improve results. Heat-shock protein induction may confer chondroprotection. The expression of heat-shock proteins in human chondrocytes and rat femoral head cartilage following heat shock was analysed

by Western blotting, and red-blood-cell-induced chondrocyte death was assessed by cell viability and apoptosis by flow cytometry. We demonstrate that heat-shock induced expression of heat-shock protein 70 (HSP70) (rat and human) and HSP32 (human). Blood and blood products reduced rat cartilage proteoglycan synthesis and human chondrocyte viability, and induced human chondrocyte apoptosis at concentrations considerably lower than those reported previously. The induction of HSP70 in rat cartilage was ineffective in reducing chondrocyte death in the absence or presence of red blood cells or red cell products. Heat shock to human chondrocytes reduced low levels of apoptosis (<20%) and cell death induced by low levels of blood products, but not higher levels. Induction of HSP32 with diacetylrhein appeared to be more effective and may hold greater promise. Blood has potent adverse effects on chondrocytes and the induction and chondroprotective effects of heat-shock proteins could be applied to increase the initial success of implanted chondrocytes improving the outcome of autologous chondrocyte transplantation.

L6 ANSWER 9 OF 9 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
ACCESSION NUMBER: 1950:33916 BIOSIS Full-text
DOCUMENT NUMBER: PREV19502400034082; BA24:34082
TITLE: Results of thr trials with growth substances on vines made since 1937 in the Palatinate.
Original Title: Ergebnisse der seit 1937 in der **Rhein**-pfalz durchgefuhrten Wuchsstoffversuche an Reben.
AUTHOR(S): KORDES, H.
SOURCE: WEIN U REBE, (1943) Vol. 25, No. 7/9, pp. 116-121.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: Unavailable
ENTRY DATE: Entered STN: May 2007
Last Updated on STN: May 2007

AB Treatments with growth hormones should be applied: during grafting to promote growth between parent stock and **graft**; immediately before setting of the vines into the nursery to improve root production; and after pruning the roots immediately before planting in the vineyard. ABSTRACT AUTHORS: F. Schwanitz

=> d hist

(FILE 'HOME' ENTERED AT 11:05:15 ON 24 JUL 2007)

FILE 'CAPLUS, MEDLINE, BIOSIS' ENTERED AT 11:05:36 ON 24 JUL 2007
L1 244960 S TRANSPLANT
L2 428898 S GRAFT
L3 434 S RHEIN/CN

FILE 'REGISTRY' ENTERED AT 11:06:22 ON 24 JUL 2007
L4 1 S RHEIN/CN
L5 1 S DIACEREIN/CN

FILE 'CAPLUS, MEDLINE, BIOSIS' ENTERED AT 11:06:54 ON 24 JUL 2007
L6 9 S (L4 OR RHEIN OR L5 OR DIACEREIN) AND (L1 OR L2)
L7 973953 S INFLAMM?

=> s (L4 OR RHEIN OR L5 OR DIACEREIN)
L8 2321 (L4 OR RHEIN OR L5 OR DIACEREIN)

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=> s l7 and l8
L9          249 L7 AND L8

=> duplicate
ENTER REMOVE, IDENTIFY, ONLY, OR (?):remove
ENTER L# LIST OR (END):19
DUPLICATE PREFERENCE IS 'CAPLUS, MEDLINE, BIOSIS'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n
PROCESSING COMPLETED FOR L9
L10         193 DUPLICATE REMOVE L9 (56 DUPLICATES REMOVED)

=> d ibib abs 180-193

L10  ANSWER 180 OF 193  CAPLUS  COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER:         1989:445276  CAPLUS Full-text
DOCUMENT NUMBER:          111:45276
TITLE:                    Oral pharmaceuticals containing rhein
                          derivatives for delayed release
INVENTOR(S):              Springolo, Vanna; Coppi, Germano; Scevola, Mario
                          Ercole
PATENT ASSIGNEE(S):       Proter S.p.A., Italy
SOURCE:                   Eur. Pat. Appl., 6 pp.
                          CODEN: EPXXDW
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.           KIND   DATE        APPLICATION NO.          DATE
     ---------------      ----   --------    --------------------     --------
     EP 264989             A1    19880427    EP 1987-201845           19870925
     EP 264989             B1    19911218
         R:  BE, CH, DE, FR, GB, LI
     JP 63146816           A     19880618    JP 1987-244417           19870930
     JP 2572781            B2    19970116
     US 4861599            A     19890829    US 1987-102936           19870930
PRIORITY APPLN. INFO.:                        IT 1986-21867       A    19861001
AB   Slow-release granules comprise rhein or a deriv. thereof, and a film coating
     compound selected from a group consisting of PVP, shellac, hydroxypropyl Me
     cellulose, and their mixts.  Diacetylrhein 500 g were mixed with PVP 10,
     microcryst. cellulose 50, and Na citrate 100 g; the mixture was wet granulated
     with water and the granulates were dried.  The granules were combined with
     granules containing soya polysaccharides 125, talc 10, and Mg stearate 150 g
     and tableted.  The tablets were coated by application of EtOH solution
     containing hydroxypropyl Me cellulose 40, acetylated monoglyceride 10, and
     TiO2 10 g.  The tablet released in vitro 40.7, 46.7, 54.7, 68.5, and 80.3% of
     diacetylrhein at 1, 4, 8, 18, and 24 h, resp.


L10  ANSWER 181 OF 193     MEDLINE on STN
ACCESSION NUMBER:    88326385     MEDLINE Full-text
DOCUMENT NUMBER:     PubMed ID: 3415721
TITLE:               Effect of diacetylrhein on the phagocytosis of
                     polymorphonuclear leucocytes and its influence on the
                     biosynthesis of hyaluronate in synovial cells.
AUTHOR:              Schongen R N; Giannetti B M; van de Leur E; Reinards R;
                     Greiling H
CORPORATE SOURCE:    Institut fur Klinische Chemie und Pathobiochemie,
                     Medizinischen Fakultat Rheinisch-Westfalischen, Technischen
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Hochschule Aachen, (Fed. Rep. of Germany).
SOURCE: Arzneimittel-Forschung, (1988 May) Vol. 38, No. 5, pp. 744-8.
Journal code: 0372660. ISSN: 0004-4172.
PUB. COUNTRY: GERMANY, WEST: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198810
ENTRY DATE: Entered STN: 8 Mar 1990
Last Updated on STN: 8 Mar 1990
Entered Medline: 11 Oct 1988

AB 1. The influence of diacetylrhein on the luminol-induced chemiluminescence of zymosan-activated polymorphonuclear leucocytes (PMNL) was investigated. At a concentration of 4 x 10(-5) mol/l diacetylrhein an inhibition of about 40% was found. 2. A model for the degradation of hyaline cartilage by frustrated phagocytosis was developed, in which human polymorphonuclear leucocytes cause a release of glycosaminoglycan peptides from hyaline cartilage slices (bovine nasal septum). We observed a 20% inhibition of this release at a concentration of 10(-4) mol/l diacetylrhein. 3. Human synovial fibroblasts synthesize the glycosaminoglycan hyaluronate. As a parameter of the rate of hyaluronate synthesis we measured the incorporation of 14C-glucosamine into hyaluronate. At a concentration of 2 x 10(-4) mol/l diacetylrhein a 4-fold increase of 14C-glucosamine incorporation in the membrane fraction of the synovial cells (tryptic fraction) and a 1.6-fold elevation of glucosamine release into the medium was measured. The synovial fibroblasts show a higher (1.5-fold) glucose consumption and lactate production in the presence of diacetylrhein (2 x 10(-4) mol/l).

L10 ANSWER 182 OF 193 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1989:128415 CAPLUS Full-text
DOCUMENT NUMBER: 110:128415
TITLE: Inhibitors of bacterial collagenase in Rhei Rhizoma
AUTHOR(S): Mineo, Satoshi; Tanaka, Toshiaki; Metori, Koichi; Niyino, Yasunori; Matsumoto, Hitoshi; Sato, Toshio
CORPORATE SOURCE: Dep. Pharmacol., Kohno Clin. Med. Res. Inst., Tokyo, 140, Japan
SOURCE: Shoyakugaku Zasshi (1988), 42(3), 249-51
CODEN: SHZAAY; ISSN: 0037-4377
DOCUMENT TYPE: Journal
LANGUAGE: English

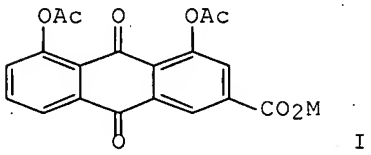
AB A hot aq. ext. of Rhei Rhizoma had an inhibitory effect on the bacterial collagenase from Clostridium histolyticum. Powdered Rhei Rhizoma was treated with organic solvents. Emodin was obtained from the CHCl3 extract as an inhibitor of the collagenase. The concentration of emodin in the assay mixture required to give 50% inhibition (IC50) was 4.0 + 10-5M, which was smaller than that of tetracycline or N-acetyl-L-cysteine. Of the reported anthraquinones from Rhei Rhizoma, aloe-emodin and **rhein** showed inhibitory effects, while sennoside A, B and aloin did not show any inhibitory effect. Acetylated products of emodin and aloe-emodin had no inhibitory activity.

L10 ANSWER 183 OF 193 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1988:26988 CAPLUS Full-text
DOCUMENT NUMBER: 108:26988
TITLE: Diacetylrhein salts for parenteral administration, and their use in the treatment of arthritis
INVENTOR(S): Dall'Asta, Leone; Coppi, Germano; Scevola, Mario Ercole

PATENT ASSIGNEE(S): Proter S.p.A., Italy
SOURCE: Eur. Pat. Appl., 9 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 243968	A2	19871104	EP 1987-106287	19870430
EP 243968	A3	19880120		
EP 243968	B1	19910821		
R: BE, CH, DE, FR, GB, LI				
JP 63008354	A	19880114	JP 1987-106538	19870501
JP 2636847	B2	19970730		

PRIORITY APPLN. INFO.: IT 1986-20298 A 19860502
OTHER SOURCE(S): MARPAT 108:26988
GI



I

AB Diacetylrhein salts I (M = alkali, alk. earth metal, org. base), which are useful against arthritis, especially osteoarthritis, are prepared for parenteral use. I (M = H) suspended in Me2CO-H2O was treated with Et3N to give a clear solution, to which was added K 2-ethylhexanoate in iso-BuOH-Me2CO to give crystalline I (M = K). I (M = Na) and I (M = K), at 2 mg/kg i.p. daily for 7 wk, reduced retinoic acid-induced arthritic deformation in rabbits by 50.5 and 65.3%, resp.

L10 ANSWER 184 OF 193 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 34
ACCESSION NUMBER: 1989:147401 CAPLUS Full-text
DOCUMENT NUMBER: 110:147401
TITLE: Experimental studies on diacerhein: effects on phagocytosis by neutrophil cells from subcutaneous carrageenan-induced exudate
AUTHOR(S): Mian, M.; Trombi, L.; Rosini, S.; Benetti, D.; Caracciolo, F.; Carulli, G.; Azzara, A.; Ambrogi, F.
CORPORATE SOURCE: Inst. Gentili S.p.A., Pisa, Italy
SOURCE: Drugs under Experimental and Clinical Research (1987), 13(11), 695-8
CODEN: DECRDP; ISSN: 0378-6501
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Diacerhein (DAR) (I), a new drug which is particularly suitable for the treatment of osteoarthritis, was studied for its interference with the phagocytic capacity of cells coming from exudates of s.c. carrageenan edema and from the peripheral blood of Sprague-Dawley rats. DAR inhibited phagocytosis in both types of cells examined This finding indicates that DAR

may exert its action by means of a direct effect on the cells involved in the **inflammatory** process.

L10 ANSWER 185 OF 193 MEDLINE on STN
ACCESSION NUMBER: 89029650 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 2972506
TITLE: [Drug therapy of arthrosis].
La terapia farmacologica dell'artrosi.
AUTHOR: Cervini C; Grassi W
SOURCE: La Clinica terapeutica, (1987 Dec 31) Vol. 123, No. 6, pp. 493-8. Ref: 27
Journal code: 0372604. ISSN: 0009-9074.
PUB. COUNTRY: Italy
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: Italian
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198812
ENTRY DATE: Entered STN: 8 Mar 1990
Last Updated on STN: 8 Mar 1990
Entered Medline: 22 Dec 1988

L10 ANSWER 186 OF 193 MEDLINE on STN
ACCESSION NUMBER: 87173683 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 3561852
TITLE: [Endoscopic evaluation of the effects of diacerhein and naproxen on the gastroduodenal mucosa].
Valutazione endoscopica degli effetti della diacereina e del naprossene sulla mucosa gastroduodenale.
AUTHOR: Bianchi Porro G; Ardizzone S; Caruso I; Montrone F
SOURCE: Minerva medica, (1987 Mar 31) Vol. 78, No. 6, pp. 411-3.
Journal code: 0400732. ISSN: 0026-4806.
PUB. COUNTRY: Italy
DOCUMENT TYPE: (COMPARATIVE STUDY)
(ENGLISH ABSTRACT)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: Italian
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198705
ENTRY DATE: Entered STN: 3 Mar 1990
Last Updated on STN: 3 Mar 1990
Entered Medline: 14 May 1987

AB The potential capacity of diacerheine (DAR), a new drug known to have no anti-prostaglandin effect and therefore a different action mechanism from the standard non-steroid anti-**inflammatory** drugs, to cause damage to the gastroduodenal mucosa was compared with that of a well-known NSAID, naproxene. Gastroscopic examination of 10 + 10 patients before and 4 weeks after treatment showed that DAR produced endoscopic lesions without subjective symptoms in 10% of the patients whereas naproxene produced lesions of varying size in 50%, with or without symptoms.

L10 ANSWER 187 OF 193 MEDLINE on STN
ACCESSION NUMBER: 87039830 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 3774205
TITLE: [Influence of diacereine on the gastroduodenal mucosa of ulcer patients in remission and on the diuresis of cirrhosis ascites patients].
Influenza della diacereina sulla mucosa gastroduodenale

degli ulcerosi in fase di remissione e sulla diuresi del cirrotico ascitico.
AUTHOR: Grimoldi D; Bellati G; Fesce E; Ideo E
SOURCE: Minerva medica, (1986 Nov 10) Vol. 77, No. 42-43, pp. 1989-95.
Journal code: 0400732. ISSN: 0026-4806.
PUB. COUNTRY: Italy
DOCUMENT TYPE: (ENGLISH ABSTRACT)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: Italian
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198612
ENTRY DATE: Entered STN: 2 Mar 1990
Last Updated on STN: 2 Mar 1990
Entered Medline: 18 Dec 1986

AB Diacereine (DAR) is a new anti-arthrosis drug with an unusual action mechanism. Once it was found that, unlike existing FANS, DAR has no effect on prostaglandin synthesis, it was thought interesting to assess its clinical tolerability on two groups of patients. The first was a group of high risk patients with arthrosis or arthritis. The second consisted of patients with a history of duodenal ulcer or cirrhotics in the ascitic phase. In the first group the DAR was given (100 mg per diem per os) for 30 days after oesophagogastroduodenoscopy that was repeated at the end of treatment. DAR treatment of patients with ulcers in the clinical remission phase was reliable in the sense that no recurring ulceration or major endoscopic lesions were observed but the subjective tolerability was not excellent in about half the patients due to the appearance of dyspeptic symptoms that are, however, a notoriously common response to any drug treatment in this kind of case series. In a second group of 5 patients with various types of cirrhosis of the liver, the aim was to assess the effect of DAR treatment on kidney function and ascitic decompensation. The drug was administered orally in doses of 100 mg per diem for 10 days. On the basis of the results observed in this case series only it can be stated that the use of DAR on ascitic cirrhosis patients produces no alterations in kidney function and does not reduce the effectiveness of diuretic treatment. Indeed it may well be that DAR has a positive effect on diuresis, that increased in this series in line with earlier experimental results.

L10 ANSWER 188 OF 193 MEDLINE on STN
ACCESSION NUMBER: 90373360 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 3079362
TITLE: **[Diacerein]**.
La diacereina.
AUTHOR: Passiu G
SOURCE: Annali italiani di medicina interna : organo ufficiale della Societa italiana di medicina interna, (1986 Jun) Vol. 1, No. 2, pp. 172-4. Ref: 9
Journal code: 8806705. ISSN: 0393-9340.
PUB. COUNTRY: Italy
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: Italian
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199010
ENTRY DATE: Entered STN: 22 Nov 1990
Last Updated on STN: 22 Nov 1990
Entered Medline: 15 Oct 1990

L10 ANSWER 189 OF 193 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on

STN

ACCESSION NUMBER: 1985:391444 BIOSIS Full-text
DOCUMENT NUMBER: PREV198580061436; BA80:61436
TITLE: DIACETYLRHEIN IN THE MANAGEMENT OF DEGENERATIVE ARTHROPATHIES.
AUTHOR(S): ADAMI S [Reprint author]; BORTOLOTTI R; GUARRERA G; MARINI G; ROSINI S; ZAMPIERI A; LO CASCIO V
CORPORATE SOURCE: CATTEDRA E REPARTO DI SEMEIOTICA MED, DELL'UNIV DI VERONA
SOURCE: Clinica Terapeutica, (1985) Vol. 112, No. 5, pp. 439-444. CODEN: CLTEA4. ISSN: 0009-9074.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ITALIAN

AB Diacetylrhein (DAR) was given to 50 patients with osteoarthritis (50 mg b.i.d. [2 times a day]) for 2 wk followed by 50 mg daily for 2-8 wk. Response to treatment was assessed by pain score, joint function (Lee's index) and analgesic consumption. The results were classified as excellent or good in 80% of the patients. Improvement was usually apparent 6-10 days after starting treatment and remission lasted about 2 wk after drug withdrawal. Increased frequency of defecation and abdominal cramps, the only minimal side effects (50% of patients), were usually well tolerated and related to laxative properties of the compound. The action of DAR is not related to inhibition of prostaglandin synthetase activity and the gastric tolerance is excellent. DAR may represent an alternative to the more common non-steroidal anti-**inflammatory** agents.

L10 ANSWER 190 OF 193 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 35
ACCESSION NUMBER: 1985:4330 CAPLUS Full-text
DOCUMENT NUMBER: 102:4330
TITLE: DNA changes in spinal cords of rats with experimental allergic encephalomyelitis
AUTHOR(S): Smith, Marion Edmonds; Somera, F. Paul; Saldivar, Robert; Massacesi, Luca; Trotter, Jacqueline
CORPORATE SOURCE: Dep. Neurol., Veterans Adm. Med. Cent., Palo Alto, CA, 94304, USA
SOURCE: Journal of Neurochemistry (1984), 43(6), 1635-41 CODEN: JONRA9; ISSN: 0022-3042
DOCUMENT TYPE: Journal
LANGUAGE: English

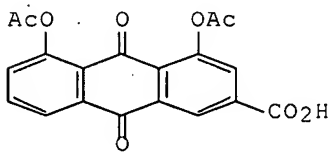
AB DNA levels were measured in the spinal cords of Lewis rats during the development of and recovery from exptl. allergic encephalomyelitis (EAE) (multiple sclerosis model). Spinal cord DNA was first increased 11 days after immunizing the rats with guinea pig myelin and rose to levels 4 times that of the Freund's adjuvant controls at day 14, then subsided after day 22. Spinal cord DNA was still 150% of control levels 60 days after immunization. These DNA changes were compared with fluctuations in spinal cord acid proteinase in the same animals. Acid proteinase activity in EAE spinal cord increased later than the rise in DNA and attained a level of 170% of control at days 15-17, then subsided. Spinal cord DNA was higher in rats immunized with whole myelin than in those administered equivalent amts. of purified myelin basic protein. Furthermore DNA was higher in spinal cords of rats immunized with a larger dose of myelin (1.0 mg) than with a lower amount (0.5 mg). Various protease inhibitors including pepstatin, nitrophenyl p-guanidino benzoate, polylysine, and dipropionyl **rhein**, previously shown to protect Lewis rats against EAE, suppressed the increase of DNA in the spinal cord. Measurement of DNA increases in the spinal cord of EAE animals provides a convenient reproducible measurement of the severity of **inflammation** in the central nervous system and provides an objective criterion for assessment of the efficacy of various agents screened as possible therapeutic treatments for multiple sclerosis.

L10 ANSWER 191 OF 193 MEDLINE on STN
ACCESSION NUMBER: 83215755 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 6133942
TITLE: The influence of **rhein** on the biosynthesis of prostaglandin-like substances in-vitro.
AUTHOR: Franchi-Micheli S; Lavacchi L; Friedmann C A; Zilletti L
SOURCE: The Journal of pharmacy and pharmacology, (1983 Apr) Vol. 35, No. 4, pp. 262-4.
Journal code: 0376363. ISSN: 0022-3573.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: (IN VITRO)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198307
ENTRY DATE: Entered STN: 19 Mar 1990
Last Updated on STN: 6 Feb 1995
Entered Medline: 8 Jul 1983

L10 ANSWER 192 OF 193 CAPLUS COPYRIGHT 2007 ACS on STN
ACCESSION NUMBER: 1982:622966 CAPLUS Full-text
DOCUMENT NUMBER: 97:222966
TITLE: Carboxy anthraquinones for treatment of arthritis
INVENTOR(S): Friedmann, Charles A.
PATENT ASSIGNEE(S): Italy
SOURCE: U.S., 6 pp. Cont. of U.S. Ser. No. 112,824, abandoned.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
US 4346103	A	19820824	US 1981-264817		19810518
ZA 7601627	A	19780125	ZA 1976-1627		19760316
US 4244968	A	19810113	US 1977-773406		19770301
PRIORITY APPLN. INFO.:			ZA 1976-1627	A	19760316
			US 1977-773406	A2	19770301
			US 1980-112824	A1	19800117

OTHER SOURCE(S): MARPAT 97:222966
GI



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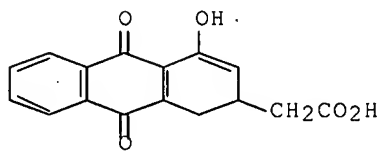

II

AB Anthraquinones contg. OH, NH2, or ester groups, and solubilizing CO2H groups, are used in the treatment of arthritis or multiple sclerosis. The effectiveness of diacetylrhein (I) [13739-02-1] was demonstrated in patients with rheumatoid arthritis. II [65175-63-5] was prepared by acetylating 1-hydroxy-3,4-dihydroanthraquinone [65175-76-0], brominating the 1-acetoxy

derivative [65175-77-1], treating the 3-bromo derivative [65929-77-3] with BrCH2CO2Et [105-36-2] and Cu powder and hydrolyzing the resulting Et 1-acetoxy-3-carboxymethyl-3,4- dihydroanthraquinone ester [65175-78-2].

L10 ANSWER 193 OF 193 MEDLINE on STN
ACCESSION NUMBER: 81090886 MEDLINE Full-text
DOCUMENT NUMBER: PubMed ID: 7450019
TITLE: A non steroidal anti-**inflammatory** drug that stimulates prostaglandin release.
AUTHOR: Pomarelli P; Berti M; Gatti M T; Mosconi P
SOURCE: Il Farmaco; edizione scientifica, (1980 Oct) Vol. 35, No. 10, pp. 836-42.
Journal code: 0370716. ISSN: 0430-0920.
PUB. COUNTRY: Italy
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198103
ENTRY DATE: Entered STN: 16 Mar 1990
Last Updated on STN: 16 Mar 1990
Entered Medline: 24 Mar 1981

AB Diacetylrhein (DAR) is a new anti-**inflammatory** and anti-osteoarthritic drug. Studies with isolated lung preparation showed that DAR does not exert its action by inhibiting the arachidonic acid metabolism. Furthermore, the in vivo experiments showed that DAR, contrary to most anti-**inflammatory** drugs, induced an increase of prostaglandin-like substances in the rat exudates. The above results are substantiated by experimental evidence that in the rat this compound displays a dose-dependent protecting activity against indomethacin-induced gastric damage.